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Bile acid malabsorption after continent urinary diversion with an ileal reservoir.

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PURPOSE: We determine the effect of urinary diversion with a Kock ileal reservoir on bile acid absorption and bowel habits. MATERIALS AND METHODS: We asked 96 patients with a Kock ileal urinary reservoir to record bowel habits and abdominal symptoms for 1 week. Data on 75 patients were further analyzed. Bile acid absorption was determined in 29 healthy control subjects, in 17 before and 6 months after continent urinary diversion, and in 21, 2 to 14 years postoperatively. Bile acid absorption was considered pathological when retention of less than 10% of an oral capsule containing selenium-75 labeled tauroselcholic acid (SeHCAT) was noted after 1 week. RESULTS: Mean number of defecations plus or minus standard deviation was 9.4 +/- 6.1 (75 cases). Of the patients 13% had 15 or more stools per week and 15% complained of always having loose stools. Mean value for the SeHCAT test was 32 +/- 19% preoperatively and 17 +/- 16% 6 months postoperatively ($p = 0.0023$). The corresponding value for healthy controls was 39 +/- 18%. Significant relationships were found between the results of the SeHCAT test postoperatively, and the number of stools per week and consistency of the feces. All patients with more than 10 defecations per week had a pathological SeHCAT test. CONCLUSIONS: Most patients with an ileal urinary reservoir have fairly normal bowel habits. Bile acid absorption is significantly reduced postoperatively and approximately a third of the patients have a pathological SeHCAT test. Preoperative investigation of bowel habits is recommended and a SeHCAT test should be performed in patients with frequent, loose defecations. Other types of diversion should be offered when preoperative retention is below 10 to 20% especially in patients with impaired anal control.

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1: Biosci Biotechnol Biochem. 1995 Apr;59(4):624-7.

Free bile acids inhibit IgE production by mouse spleen lymphocytes stimulated by lipopolysaccharide and interleukins.

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The interaction of IL-4, IL-5, and free bile acids with the immunoglobulin production by mouse spleen lymphocytes was studied to examine their immunoregulatory activity. In the absence of lipopolysaccharide (LPS), IL-4 enhanced the IgE and IgG production significantly and the IgA production weakly, but not the IgM production. On the other hand, IL-5 had an inhibitory tendency on the IgE and IgA production, though not significantly. In the presence of LPS, both IL-4 and IL-5 significantly enhanced the IgE production by mouse splenic lymphocytes. When the lymphocytes were cultured with the physiological concentration of free bile acids (10 microM) and LPS for 3 days, chenodeoxycholic acid inhibited the IgE production, but cholic and deoxycholic acids did not. In the presence of IL-4 or IL-5, these bile acids cancelled the stimulatory effects of interleukins and rather significantly inhibited the IgE production. These results suggest that these free bile acids act as an anti-allergic agent.

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Ursodeoxycholic acid modifies gut-derived endotoxemia in neonatal rats.

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We developed a model for the translocation of intraluminal endotoxin in the neonatal animal and used it to examine the capacity of a nonhepatotoxic bile acid, ursodeoxycholic acid (UDCA), to modify endotoxin translocation and cytokine response. Three-d-old Sprague-Dawley rats were randomized to receive enterally either no drug, lipopolysaccharide (LPS, 1 mg/animal), or UDCA (400 micrograms/animal) alone, or UDCA followed by LPS 1 h later. One h after LPS administration, the rats were killed and plasma endotoxin and tumor necrosis factor (TNF) were measured. Control animals had low circulating endotoxin (21.2 +/- 7.6 endotoxin units) and TNF (0.06 +/- 0.02 ng/mL). Enteral administration of LPS 1 h before the rats were killed resulted in significant elevation of endotoxin (249.5 +/- 71.3, p = 0.008) and TNF (3.6 +/- 1.3, p = 0.019). UDCA alone did not alter endotoxin levels (8.7 +/- 2.1). UDCA 1 h before LPS prevented the rise in endotoxin (38.9 +/- 11.2 endotoxin units) and TNF (0.2 +/- 0.05) significantly. Chenodeoxycholic acid was studied in a similar group of experiments and prevented neither the translocation of LPS nor the development of increased TNF levels in animals receiving LPS. In conclusion, LPS can cross the intestinal barrier in the normal neonatal rat. UDCA, administered before LPS, can decrease the translocation of LPS and prevent the cytokine response as measured by TNF levels. We speculate that UDCA, administered prophylactically, might reduce morbidity in clinical conditions leading to gut-derived endotoxemia.

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